Treating Carbon Monoxide Poisoning – Is Continuous Positive Airway Pressure Advantageous over Oxygen Therapy?

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Respected Editor,

Carbon monoxide (CO), a leading cause of poisoning, causes its noxious effects by depriving the living tissues of Oxygen (O2). It does so by binding to hemoglobin (Hb) with a significantly higher affinity than O2. The resulting Carboxyhemoglobin (COHb) decreases the blood's O2 carrying capacity and even impairs the release of O2 from Hb [1]. As organs' vitality becomes progressively compromised, the symptoms of CO poisoning aggravate from vague complaints such as headache, dizziness, blurred vision, nausea and vomiting to hypotension, arrhythmias, cardiac and respiratory arrest, seizures, and ultimately coma [1]. The most toxic amounts of CO typically exist in indoor air. Wood burning stoves, furnaces, inappropriately adjusted gas appliances, improperly vented gas generators inside a house or building are potential contributors to CO poisoning [2].

Oxygen therapy delivered *via* a non-rebreather mask or endotracheal tube has historically been the mainstay of treatment in CO poisoning. Hyperbaric Oxygen therapy has also been suggested, especially in severe cases, with evidence supporting the fact that it lowers the half-life of CO faster than normobaric oxygen. However, its use is severely limited due to cost constraints, lack of expertise, and the undersupply of equipment, even more so in the developing world [1]. A relatively new approach is being investigated, which involves delivering continuous positive airway pressure (CPAP) through non-invasive mechanical ventilation [3-5]. The resulting positive pressure in the airways improves the exchange of gases and leads to a rise in alveolar pressure. The increased inspiratory and expiratory pressures in the alveoli, in turn, have a favorable impact on the ventilation/perfusion ratio, which is hypothesized to cause an increase in the partial pressure of O2, increased O2 binding with hemoglobin, and CO decomposition [4].

Turgut *et al.* [3] compared the lowering of CO between patients given 15 L/min O2 *via* non-rebreather mask and patients given FiO2 100% *via* CPAP. In this study, they observed a statistically significant lowering of CO at 30, 60, and 90 minutes with CPAP compared to normobaric O2. Furthermore, the duration of stay in the Emergency department decreased from a median of 185 minutes to 125 minutes in normobaric Oxygen and CPAP groups, respectively. The half-life of CO was also lowered from a median of 105 minutes in the normobaric oxygen group to 45 minutes in the CPAP group [3]. Another study by Bal *et al.* [4] comparing the efficacy of 10 L/min O2 vs. CPAP found a similar decline in the half-life of COHb using CPAP *i.e.* 80.26 ± 12.70 minutes for O2 therapy *vs.* 36.20 ± 4.58 minutes for CPAP. As obtained by venous samples, the blood CO levels were also significantly lower in the CPAP group at 30 and 60 minutes [4]. Caglar *et al.* [5] further reported that CPAP was superior to high-flow O2 therapy to lower the COHb saturation within the first 30 min of treatment. The median lowering of COHb was 13% in the CPAP group and 6% in the patients on O2 therapy, as shown by the CO-oximeter. However, both treatment modalities provided approximately similar relief of symptoms such as headache and nausea, with CPAP showing a slight benefit in lowering headaches [5].

The studies mentioned above are, however, limited by their small sample sizes. Thus, large-scale studies are needed to establish the role of CPAP in acute CO poisoning. Since the trials largely excluded vitally unstable patients with complications and high blood CO levels, no effective conclusion can be drawn regarding CPAP's efficacy in severe CO poisoning. Sufficient data to suggest its impact on mortality and long-term neurocognitive effects is also lacking. Thus, these areas require further investigation. Despite these gaps, we can conjecture that the use of CPAP may facilitate a rapid lowering of COHb levels and, quite possibly, better symptom control and earlier discharge from the emergency department.

CONFLICT OF INTEREST

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

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Received: November 24, 2020; Revised: January 01, 2021; Accepted: January 02, 2021
DOI: https://doi.org/10.37184/lnjpc.2707-3521.3.1

FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

ACKNOWLEDGMENTS

Both the authors (M.S.S., Mishal Shan Siddiqui and S.K., Saad Khalid) had full access to data and contributed to the study. All authors read and approved the final version of the manuscript and take responsibility for its accuracy and integrity. M.S.S. contributed to the concept and design of the study. M.S.S. and S.K. drafted the manuscript. S.K. critically revised the manuscript.

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